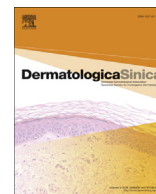


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CORRESPONDENCE

Herpes vegetans



An 82-year-old man had two asymptomatic vegetative tumors on his right groin for 1.5 years. He had a history of chronic lymphocytic lymphoma and was currently taking 2 mg chlorambucil every other day for disease control. Both tumors were eroded. One of the tumors adjacent to the scrotum was covered with a thin crust, and the other tumor on his right groin wept with serous fluid (Figure 1A–C). Owing to the suspicion of squamous cell carcinoma, an excisional biopsy of the groin lesion was performed. The pathology revealed ulceration and granulation tissue with densely and diffusely mixed cell infiltration. Multinucleated giant cells of keratinocytes, necrotic ballooning keratinocytes, and eosinophilic intranuclear inclusion bodies were noted at the base of the ulcer (Figure 2A and B). The patient was diagnosed with herpes vegetans according to the clinical and pathological presentation. Tracing back the detailed history, the patient had experienced ulcerative lesions over his right groin 2 years ago, during which herpes simplex virus type 2 was isolated at another hospital. Since then, the ulcers gradually grew into tumors over a period of 5 months, just before the patient visited our dermatology clinic. Accordingly, he was treated with oral famciclovir (250 mg) three times daily and topical 1% tromantadine serol, and the other tumor near the scrotum shrank and dried up.

Herpes vegetans is a rare variant of cutaneous herpes simplex virus infection. It is also known as chronic hypertrophic herpes or chronic herpes simplex infection.¹ Herpes virus infection is a common cutaneous disease; however, the presentation may be atypical in immunocompromised patients, thereby making the diagnosis difficult. Several unusual clinical features have been

described, including generalized papular eruption, hyperkeratotic verrucous lesions, and erosive vegetating plaques.¹

Although herpes vegetans is mostly described in patients with human immunodeficiency virus infection (HIV)/AIDS,^{2–4} it can also occur in patients with other types of immunodeficiency, such as common variable immunodeficiency, hematologic malignancy, congenital immunodeficiency, and bone marrow transplantation. The lesions are usually located on genitocrural areas, such as vulva, penis, scrotum, inguinal, and perianal areas. Occasionally, it may appear on the tongue and eyelids. The differential diagnosis of these verrucous and ulcerative nodules on groins and genital areas includes condylomata accuminata, condylomata lata, severe hyperplastic candidiasis, mycobacterial and fungal infection, extramammary Paget's disease, pyogenic granuloma, keratoacanthoma, verrucous carcinoma, and squamous cell carcinoma. Viral cultures from the ulcerated lesions may be negative despite repeated sampling. Previous reports have shown that skin biopsy remains the preferred method for correct diagnosis. Histopathologically, keratinocytes with cytopathic signs including grayish hue of the nuclei and Cowdry type A intranuclear inclusions can occasionally be identified. Confirmation of viral infection can subsequently be processed via immunohistochemical stain, *in situ* hybridization, or polymerase chain reaction methods.¹

Similar lesions may occur in varicella zoster virus infection.⁵ However, the pathogenesis of either herpes vegetans or verrucous zoster lesions remains undetermined. It has been suggested that the epidermal hyperplasia is due to cytokine release in relation to long-term infection.⁵ A genetic predisposition to the development

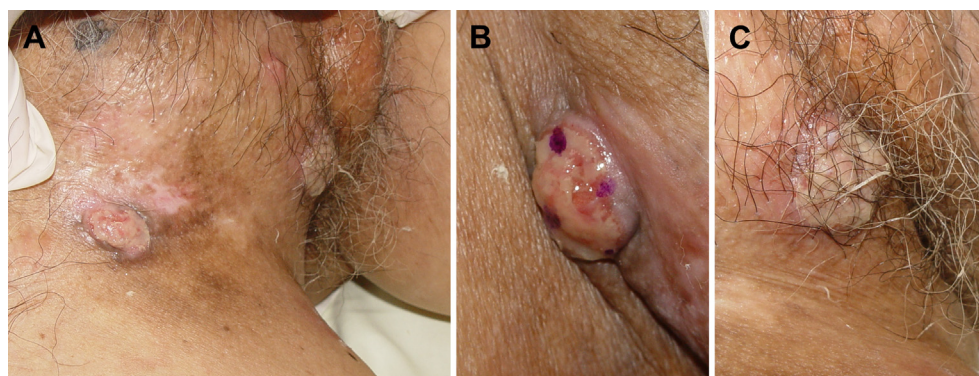


Figure 1 (A) Two vegetative tumors on the right genitocrural area. (B) The upper one at the right groin is erosive and weeping. (C) The other tumor close to the scrotum is ulcerative and covered with crust.

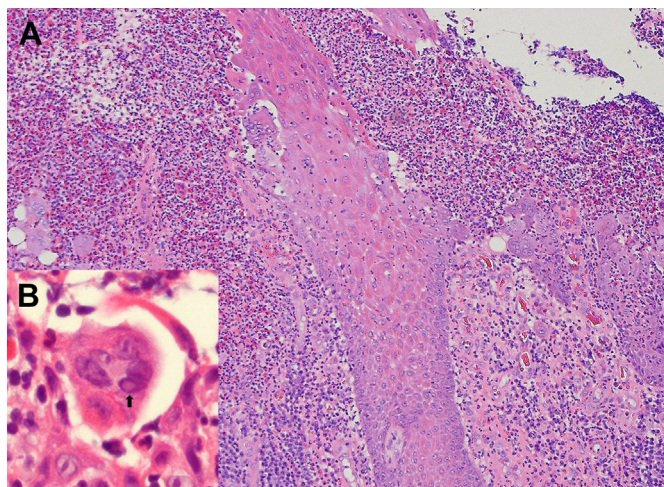


Figure 2 (A) Ulceration and granulation tissue with dense neutrophil and eosinophil infiltration (hematoxylin and eosin, 40 \times). (B) Higher magnification view shows multinucleated giant cells of keratinocyte and intranuclear eosinophilic Cowdry type A inclusion body (arrow) (hematoxylin and eosin, 400 \times).

of hypertrophic lesions has also been proposed.⁶ The hypotheses trying to explain the persistent lesions in immunocompromised host include inadequate immune response, altered viral genome, and absence or decrease of membranous expression of viral glycoproteins to elicit host immune reactions.⁵

The first line of treatment for herpes vegetans are thymidine kinase (TK)-dependent antiviral agents, although resistance to TK-dependent antiviral agents had been reported frequently.⁷ Oral acyclovir can be prescribed initially. However, in the case of unresponsiveness to acyclovir, one may either increase the dose of acyclovir to overcome bioavailability issues or switch to valacyclovir or famciclovir. In severe or extensive lesions, intravenous acyclovir should be considered. If the response to TK-dependent antiviral agents is poor, non-TK-dependent antivirals should be initiated. These include trifluorothymidine, foscarnet, cidofovir, and vidarabine, administered either topically or intravenously.¹ The combination of topical cidofovir and foscarnet has been

reported to successfully treat an HIV-positive patient with recurrent chronic hypertrophic acyclovir-resistant genital herpes.⁸ Surgical excision offers another alternative treatment for patients with single or few small lesions.

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